Clinical outcome and morphologic analysis after endovascular aneurysm repair using the Excluder endograft

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Objective: **Long-term follow-up after endovascular aneurysm repair (EVAR) is very scarce, and doubt remains regarding the durability of these procedures. We designed a retrospective cohort study to assess long-term clinical outcome and morphologic changes in patients with abdominal aortic aneurysms (AAAs) treated by EVAR using the Excluder endoprosthesis (W. L. Gore and Associates, Flagstaff, Ariz).**

Methods: **From 2000 to 2007, 179 patients underwent EVAR in a tertiary institution. Clinical data were retrieved from a prospective database. All patients treated with the Excluder endoprosthesis were included. Computed tomography angiography (CTA) scans were retrospectively analyzed preoperatively, at 30 days, and at the last follow-up using dedicated tridimensional reconstruction software. For patients with complications, all remaining CTAs were also analyzed. The primary end point was clinical success. Secondary end points were freedom from reintervention, sac growth, types I and III endoleak, migration, conversion to open repair, and AAA-related death or rupture. Neck dilatation, renal function, and overall survival were also analyzed.**

Results: **Included were 144 patients (88.2% men; mean age, 71.6 years). Aneurysms were ruptured in 4.9%. American Society of Anesthesiologists classification was III/IV in 61.8%. No patients were lost during a median follow-up of 5.0 years (interquartile range, 3.1-6.4; maximum, 11.2 years). Two patients died of medical complications ≤30 days after EVAR. The estimated primary clinical success rates at 5 and 10 years were 63.5% and 41.1%, and secondary clinical success rates were 78.3% and 58.3%, respectively. Sac growth was observed in 37 of 142 patients (26.1%). Cox regression showed type I endoleak during follow-up (hazard ratio, 3.74; $P = .008$), original design model (hazard ratio, 3.85; $P = .001$), and preoperative neck diameter (1.27 per mm increase, $P = .006$) were determinants of sac growth. Secondary interventions were required in 32 patients (22.5%). The estimated 10-year rate of AAA-related death or rupture was 2.1%. Overall life expectancy after AAA repair was 6.8 years.**

Conclusions: **EVAR using the Excluder endoprosthesis provides a safe and lasting treatment for AAA, despite the need for maintained surveillance and secondary interventions. At up to 11 years, the risk of AAA-related death or postimplantation rupture is remarkably low. The incidences of postimplantation sac growth and secondary intervention were greatly reduced after the introduction of the low-permeability design in 2004. (J Vasc Surg 2012;56:920-8.)**

Two decades after its introduction,[1] endovascular aneurysm repair (EVAR) is established as a valid treatment option for infrarenal abdominal aortic aneurysms (AAAs). Compared with open repair, there is evidence of an early survival benefit at the expense of a higher late reintervention rate.[2-5] As long-term data become available, concerns have been raised regarding the durability of EVAR, in particular, regarding the delayed risk of sac growth and rupture after implantation.[6,7]

Several endovascular devices are available for AAA repair. However, evaluation and comparison of individual endoprosthesis is especially difficult due to the lack of device-specific reporting in published studies and to the constant introduction of improvements and new devices. This study aims to analyze long-term results and morphologic changes after EVAR using the Excluder endoprosthesis (W. L. Gore and Associates, Flagstaff, Ariz), a device marketed in Europe since 1997 without major structural modifications, except for the addition of a low-permeability expanded polytetrafluroethylene sleeve to the graft composition in 2004. Our hypothesis is that EVAR can be performed with acceptable complication rates and very low long-term AAA-related mortality using an endoprosthesis that will still be available for the foreseeable future.

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METHODS

Patient population. Patient selection and data retrieval were based on a prospectively kept database of vascular surgery patients at Erasmus University Medical Center (Rotterdam, The Netherlands). Inclusion criteria were date of surgery between January 2000 and December 2007, infrarenal AAA treatment, and implantation of an Excluder endoprosthesis. Patients with previous aortic surgery or isolated iliac aneurysms were excluded. Vital status was checked once at the end of follow-up by consult of civil registry data. All causes of death were obtained. Product codes of endografts implanted in 2004 were retrieved to determine which patients received an original design (OD) or a low-permeability (LP) Excluder.

Image acquisition and postprocessing. Computed tomography angiography (CTA) was performed according to standardized institutional protocols. Morphologic analysis and measurements were performed post hoc using dedicated U.S. Food and Drug Administration-approved postprocessing software with center lumen line (CLL) reconstruction (3Mensio Vascular 4.2 software, 3Mensio Medical Imaging BV, Bilthoven, The Netherlands). CLLs were constructed semiautomatically and followed the center of the aortic and iliac permeable lumen.

All CTA scans were analyzed preoperatively, early (<30 days, typically ≤48 hours) postoperatively, and at the last follow-up visit. For patients with complications or sac growth, all other CTAs were also analyzed. No digital records of preoperative and first postoperative CTAs were kept for 28 patients (19.4%), and these measurements were performed on hard copies. Consequently, preoperative and early postoperative sac volumes were not obtainable for these patients.

Two observers (A.J., F.G.) performed all image analysis independently, blinded to patient data. Interobserver variability was assessed in a sample of 30 patients and agreement was high for AAA diameter (R^2 linear = 0.996), neck length (R^2 linear = 0.991), and neck diameter (R^2 linear = 0.935). Aneurysm sac volume was assessed according to a previously published protocol.[8]

Definitions. Neck length was defined as the length from the lowermost renal artery to the level where the aortic diameter increases by at least 10%. Maximum diameter measurements were obtained after CLL reconstruction. Technical success was defined as successful access and deployment of an endoprosthesis, without need for open conversion, type I or III endoleaks, or significant kinking or obstruction of flow. When an unplanned endovascular procedure was necessary to obtain success, during the operation or ≤24 hours, primary assisted technical success was considered. When an unplanned open surgical procedure was necessary, this was considered secondary technical success.

Clinical success was defined as successful deployment at the intended position, without death as a result of treatment, postimplantation rupture, open conversion, type I or III endoleak, device infection or thrombosis, migration, sac growth, or device integrity failure. The distance from the lowermost renal artery to the start of the endoprosthesis was serially measured and migration calculated using the first postoperative measurement as baseline. Migration was defined as downward displacement of the device by >10 mm. A lower threshold of 5 mm was considered separately but not accounted for to determine clinical success. Sac growth was defined as a diameter increase >5 mm or volume increase >5%. Neck dilatation was considered if the difference in neck diameter was ≥2 mm.

Primary clinical success required no additional or secondary procedure. In primary assisted clinical success, a preventive intervention was deemed necessary to maintain clinical success. In secondary clinical success, such a procedure was needed to correct an established complication.

Survival outcomes considered were overall survival and freedom from AAA-related death or postimplantation rupture. Thirty-day morbidity was defined as any complication that required additional procedures or prolonged hospital stay. All definitions are according to the reporting standards for EVAR.[9]

End points. The primary study end point was clinical success. Individual components of clinical success were used as secondary end points. These were freedom from reintervention, sac growth, types I and III endoleak, migration, conversion to open repair, and AAA-related death or rupture.

Additional analysis. Technical success, early (30-day or in-hospital) outcome, neck morphology changes, and overall survival are analyzed. Serum creatinine levels were obtained before surgery, before hospital discharge, and yearly thereafter. From these, estimated glomerular filtration rates (eGFR) were calculated and compared preoperatively, early postoperatively, and at the last available follow-up.[10]

Statistical methods. Continuous variables are presented as means ± standard deviation or medians and interquartile range, as appropriate. Univariate analysis for normally distributed variables was performed using the Student *t*-tests, and for nonparametric variables, Mann-Whitney *U* tests or Kruskal-Wallis tests were used. Dichotomous variables are presented as counts and percentages and compared between groups using Pearson χ^2 statistics or the Fisher exact test, as applicable. Multivariable logistic regression analysis was used to identify independent risk factors for intraoperative type Ia endoleak, and results reported as odds ratio and 95% confidence intervals (CIs). Kaplan-Meier survival curves were used to estimate clinical success and survival, and equality between groups was compared with the log-rank (Mantel-Cox) test. Univariable and multivariable Cox proportional hazard analysis was used to identify risk factors for sac growth and results reported as hazard ratios and 95% CIs. All statistical tests were two-sided and considered significant when the *P* value was <.05. Analyses were performed using SPSS 19 software (SPSS Inc, Chicago, Ill).

Table I. Baseline characteristics

Variable[a]	*Total (n = 144)*	*OD (n = 61)*	*LP (n = 83)*
Age, years	71.6 ± 8.0	70.5 ± 8.6	72.4 ± 7.5
Male sex	127 (88.2)	55 (90.2)	72 (86.7)
Hypertension	88 (60.7)	39 (63.9)	49 (59.0)
History of CAD	47 (32.6)	15 (24.6)	32 (38.5)
Moderate/severe RD	35 (24.3)	18 (29.0)	17 (20.5)
Smoking history	84 (58.3)	36 (59.0)	48 (57.8)
COPD	35 (24.3)	16 (26.2)	19 (23.0)
CVD	13 (9.0)	7 (11.5)	6 (7.2)
S-PAD	7 (4.9)	4 (6.6)	3 (3.6)
Diabetes	14 (9.7)	7 (11.5)	7 (8.4)
ASA score ≥3	89 (61.8)	37 (60.7)	52 (62.6)
RCR index ≥2	68 (47.2)	25 (41.0)	43 (51.8)
Timing of the procedure			
Elective	122 (84.7)	51 (83.6)	71 (85.5)
Symptomatic	15 (10.4)	7 (11.5)	8 (9.6)
Ruptured	7 (4.9)	3 (4.9)	4 (4.8)
Anesthesia type			
General	54 (37.5)	26 (42.6)	28 (33.7)
Regional	76 (52.8)	29 (47.5)	47 (56.6)
Local	14 (9.7)	6 (9.8)	8 (9.6)
Neck variables			
Length, mm	28.5 (21-42)	26 (18.5-37.5)	32 (21-44)
Diameter, mm	24 (22.2-25)	24 (23-25)	24 (22-25)
Angulation,°	27 (14.2-45.7)	20 (10-37.5)	32 (15-50)
AAA variables			
Diameter, mm	60 (54-70)	59 (52.5-64)	62 (54-72)
Volume, cm^3	185 (150-291)	176 (139-267)	192 (155-292)

AAA, Abdominal aortic aneurysm; *ASA,* American Society of Anesthesiologists; *CAD,* coronary artery disease; *COPD,* chronic obstructive pulmonary disease; *CVD,* cerebrovascular disease; *LP,* low-permeability design; *OD,* original design; *RCR,* revised cardiac risk; *RD,* renal dysfunction; *S-PAD,* symptomatic peripheral arterial disease.

[a]Continuous data are shown as the mean ± standard deviation or median (interquartile range) and categoric data as number (%).

RESULTS

Study population. From 2000 to 2007, 179 patients (88.3% men) underwent EVAR for infrarenal AAAs at the Erasmus University Medical Center. Of these, 144 (80.4%) were implanted with an Excluder endoprosthesis and included in the study. The mean age was 71.6 ± 7.9 years. Baseline clinical and anatomic characteristics are detailed in Table I. The following endografts were also used at our institution during the study period: Zenith (Cook Medical Inc, Bloomington, Ind) in 27 (15.1%), Lifepath (Edwards Life Sciences, Irvine, Calif) in four (2.2%), and Talent (Medtronic, Santa Rosa, Calif) in three (1.7%).

Technical success. The primary technical success rate of the EVAR procedure was 89.6%, and the primary assisted success rate was 99.3% (Table II). One conversion to open repair was performed intraoperatively due to inadvertently low deployment. The open procedure and postoperative evolution were uneventful.

Additional intraoperative endovascular procedures were required in 14 of 144 patients (9.7%). These were due to type Ia endoleak in nine, type Ib endoleak in two, and partial occlusion of a renal artery in four. Eight additional patients were found to have intraoperative type Ia endoleak, which resolved with ballooning alone.

A larger preoperative AAA diameter ($P = .010$) and greater infrarenal neck angulation ($P = .024$) were identified as risk factors for intraoperative type Ia endoleak. Also, there was a trend toward a higher risk with greater neck diameters ($P = .055$). Other baseline anatomic characteristics did not increase risk, nor did the timing of surgery

Table II. Technical success and 30-day outcome after endovascular aneurysm repair

Outcome variable	*No. (%) (N = 144)*	*95% CI*[a]
Technical success		
Primary	129 (89.6)	83.5-93.6
Primary assisted	143 (99.3)	96.2-99.9
Intraoperative conversion to open repair	1 (0.7)	1.2-3.8
Unplanned adjunct procedures[b]	14 (9.7)	5.9-15.7
Proximal balloon-expandable stent	5 (3.5)	1.5-7.9
Proximal cuff	4 (2.8)	1.1-6.9
Renal stenting	4 (2.8)	1.1-6.9
Distal extension	2 (1.4)	0.4-4.9
In-hospital or 30-day death	2 (1.4)	0.4-4.9
Myocardial infarction	1 (0.7)	1.2-3.8
Respiratory infection	1 (0.7)	1.2-3.8
In-hospital or 30-day morbidity	31 (21.5)	15.6-28.9
Graft-related	5 (3.5)	1.5-7.9
Not graft-related	26 (18.1)	12.6-25.1

[a]Confidence intervals (*CI*) are calculated for the proportions.

[b]Two procedures were performed on the same patient.

Table III. Multivariable logistic regression analysis of risk factors for intraoperative type Ia endoleak

Variable	*HR (95% CI)*[a]	P
Neck diameter	1.255 (1.013-1.555)	.038
AAA diameter	1.033 (0.990-1.078)	.129
Infrarenal angulation	1.015 (0.989-1.042)	.252

AAA, Abdominal aortic aneurysm; *CI,* confidence interval; *HR,* hazard ratio.
[a]HRs and the 95% CIs are presented per unit increase.

(elective vs urgent). In multivariable logistic regression, only neck diameter was significantly associated with intraoperative type Ia endoleak (Table III). The presence of intraoperative type Ia endoleak or the need for adjunct intraoperative procedures to achieve proximal seal was not associated with a greater risk of secondary intervention, sac growth, or migration. This remained true after correcting for the generation of implanted endoprosthesis.

In-hospital or 30-day mortality and morbidity. In-hospital or 30-day mortality rate was 1.4% as a result of two patient deaths, one on day 4 of acute respiratory failure and one on day 5 of myocardial infarction (Table II). Both patients were treated in emergency setting due to symptomatic aneurysms.

Five graft-related complications were found in the postoperative period: three type Ia endoleaks requiring reintervention (placement of balloon-expandable stents in two patients and re-ballooning a balloon-expandable stent placed intraoperatively) and two limb occlusions requiring surgical thrombectomy and angioplasty with stent placement. Median hospital stay was 3 days (interquartile range, 2-5 days) and three patients (2.1%) were discharged to another institution.

Clinical success. Median follow-up was 5.0 years (interquartile range, 3.1-6.4; maximum, 11.2 years). No patients were lost for follow-up, and the exact cause of death was obtained for all who died. Notably, 43 patients were available for follow-up more than 6 years after the original procedure (Table IV). The estimated primary clinical success rates were 63.5% and 41.1% at 5 and 10 years, and secondary clinical success rates were 78.3% and 58.3%, respectively (Fig 1).

During the follow-up period, 39 secondary interventions were performed in 32 (22.5%) patients. Problems with the proximal sealing zone were the motif for intervention in 10 patients, comprising seven type Ia endoleaks and three with increasingly short proximal sealing. Whenever possible, the sealing zone was extended by use of a proximal cuff (n = 5) or a partially covered NuMED CP balloon-expandable stent (n = 2; Heart Medical Europe BV, Best, The Netherlands). One patient was converted to aorto-monoiliac EVAR after 4 years, and two patients underwent successful open surgical conversion, after 6 months and 10.5 years, due to type Ia endoleak. Nine patients underwent implantation of limb extensions. A type Ib endoleak was identified in four, with imaging evidence in the remaining patients showing increasingly short distal sealing (n = 4) or progression of aneurysmal dilatation distal to the endoprosthesis (n = 1).

Table IV. Clinical success and long-term outcome

Outcome variable	*No. (%)*
Clinical success	
Primary clinical success	93/144 (64.6)
Primary assisted	99/144 (68.7)
Secondary	115/144 (79.9)
Endograft migration	
>5 mm	14/142 (9.9)
>10 mm	5/142 (3.5)
Sac growth	
>5 mm in diameter	34/142 (23.9)
>5% in volume	31/116 (26.7)
Without endoleak	16/142 (11.3)
With endoleak	
Type II	12/142 (8.4)
Type I	6/142 (4.2)
Secondary endoleak	
Type Ia	7/142 (4.9)
Type Ib	4/142 (2.8)
Type II	33/142 (23.2)
Secondary interventions	
Proximal balloon-expandable stent	2/142 (1.4)
Proximal cuff	5/142 (3.5)
Distal extension	11/142 (7.7)
Open/laparoscopic AAA fenestration	5/142 (3.5)
Open/laparoscopic lumbar/IMA ligation	1/142 (0.7)
Percutaneous embolization of IMA/lumbars	4/142 (2.8)
Conversion to aortouniiliac	1/142 (0.7)
Conversion to open repair	
Elective	6/142 (4.2)
Urgent	1/142 (0.7)
Relining	2/142 (1.4)
Iliac PTA	1/142 (0.7)
Mortality (including 30 days)	68/144 (47.2)
AAA-related	Mar-68 (4.4)
Oncologic	29/68 (42.6)
Cardiovascular	16/68 (23.5)
Other	20/68 (29.4)
Other secondary graft-related complications	
Endograft limb occlusion	2/142 (1.4)
Ischemic colitis	2/142 (1.4)
Buttocks claudication	2/142 (1.4
Postimplantation rupture	1/144 (0.7)

AAA, Abdominal aortic aneurysm; *IMA,* inferior mesenteric artery; *PTA,* percutaneous transluminal angioplasty.

Postimplantation sac growth was found in 34 of 142 patients. The generation of implanted endoprosthesis (OD vs LP) was significantly associated with the risk of sac growth (Fig 2). Neck diameter and occurrence of type I endoleak during follow-up were also significant risk factors for sac growth in univariable and multivariable analysis (Table V).

Elective open surgical conversion was performed in four patients with continued sac growth, despite absence of endoleak, after 1.9, 3.5, 5.5, and 10.3 years. Three of these were implanted with the OD design model. Two patients underwent relining with less permeable endoprosthesis after 4.5 and 5.2 years, for similar reasons. These successfully

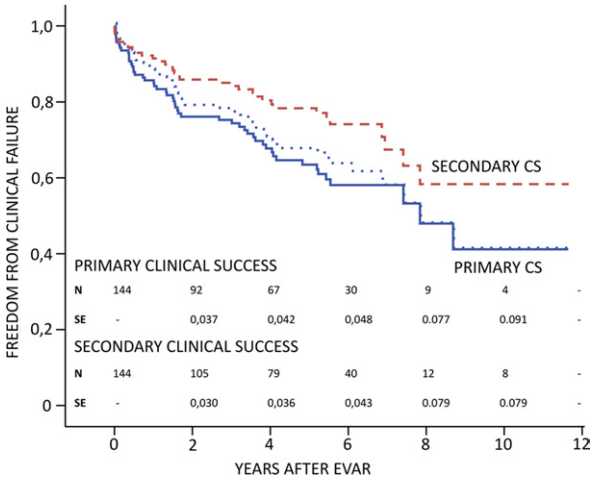

Fig 1. Kaplan-Meier estimates are shown for primary (*solid blue line*), secondary (*dashed red line*), and primary assisted (*dotted blue line*) clinical success (*CS*). *N*, Number at risk; *SE*, standard error.

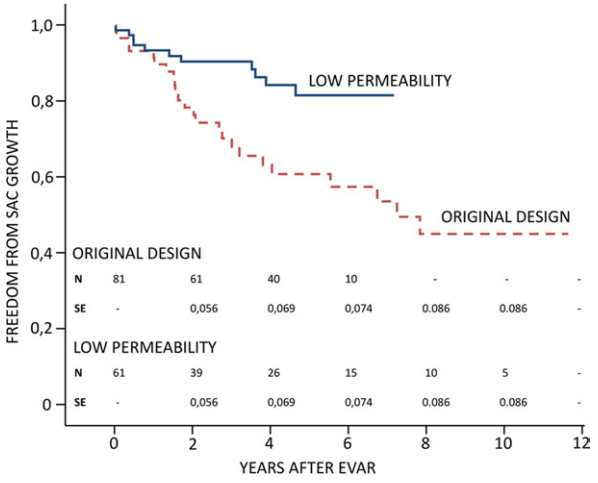

Fig 2. Kaplan-Meier estimates are shown for sac growth according to the low permeability design (*solid blue line*) and original design (*dashed red line*) of implanted endograft. $P = .005$ (log-rank test). *N*, Number at risk; *SE*, standard error.

arrested sac increase. Two patients with endotension underwent endoscopic fenestration, with similar success.

Type II endoleaks were present in 33 of 142 patients (23.2%). Eight were actively treated by means of open (n = 1) or endoscopic (n = 2) AAA sac fenestration, endoscopic lumbar ligation (n = 1), or percutaneous lumbar/inferior mesenteric artery embolization (n = 4). Indication for treatment of type II endoleak was individualized, but association to sac growth was the most common motif for treatment.

Migration ≥10 mm was observed in five patients, of which none had type I endoleak and only one required secondary intervention due to increasingly short proximal seal; however, 15 patients were identified when a lower threshold of ≥5 mm was used. Two (13.3%) of these had a type Ia endoleak and five (33.3%) underwent secondary intervention to extend seal. Univariate analysis found migration increased the risk of secondary type Ia endoleak ($P = .042$) and the need for secondary proximal neck intervention ($P = .001$). Limb occlusion was observed in two patients, of whom one underwent surgical thrombectomy, followed by percutaneous transluminal angioplasty, and the other remained asymptomatic and was managed conservatively.

Table V. A, Cox univariate regression analysis for determinants of sac growth

Variable	*HR (95% CI)*	P
Original design model	5.86 (2.77-12.36)	<.001
Endoleak during follow-up		
Type I	2.66 (1.15-6.13)	.038
Type II	1.82 (0.89-3.72)	.098
Neck angulation[a]	1.003 (0.988-1.018)	.707
Neck diameter[a]	1.185 (1.021-1.374)	.026

CI, Confidence interval; *HR*, hazard ratio.
[a]Per unit increase in endoleak.

A mean increase of 1.32 mm (95% CI, 1.05-1.58 mm) was observed between preoperative and last neck diameter, translating into a yearly growth rate of 0.24 mm. Neck dilatation was observed in 52 patients (36.6%). In 29 patients with >7 years of follow-up, neck dilatation was present in 19 (65.5%). Mean oversizing in patients with neck dilatation was 14.6% ± 6.0% vs 11.6% ± 6.6% for those without neck dilatation ($P = .008$). Proximal graft diameter did not influence dilatation. The presence of neck dilatation increased the risk of migration ≥5 mm (odds ratio, 5.5; 95% CI, 1.63-18.23), but no increased risk was found for migration ≥10 mm, occurrence of type Ia endoleak, or need for proximal neck secondary intervention. No relationship was observed between neck dilatation and sac growth.

One patient sustained rupture at 2.1 years after implantation. This patient had a shrinking aneurysm, from a 51-mm AAA diameter preoperatively to 42 mm at 2 years, without migration or other complications. At the time of rupture, there was clinical and imaging evidence of graft infection, later confirmed by positive cultures of *Staphylococcus aureus*. The patient underwent open surgery with removal of the infected prosthesis and in situ reconstruction, but died postoperatively. No other postimplantation ruptures occurred. The estimates for AAA-related death or rupture (including 30 days) were 2.4% at 5 years and 2.4% at 10 years (Fig 3).

The mean preoperative eGFR was 74.2 ± 25.9 mL/min/1.73 m^2. After surgery, a decline was observed to a mean eGFR of 69.4 ± 24.5 mL/min/1.73 m^2, which reached statistical significance ($P < .001$). However, at the last follow-up visit, the mean eGFR was 73.7 ± 30.5 mL/min/1.73 m^2, which was not significantly different from the preoperative mean value ($P = .786$).

Table V. B, Cox multivariate regression analysis for determinants of sac growth

Model	*Variable*	*HR (95% CI)*	P
Type I EL during FU	Type I EL during FU	2.848 (1.170-6.934)	.021
Plus endograft generation	Type I EL during FU	4.899 (1.870-12.830)	.001
	Original design model	3.789 (1.691-8.489)	.001
Plus neck diameter	Type I EL during FU	3.736 (1.405-9.933)	.008
	Original design model	3.849 (1.708-8.673)	.001
	Neck diameter[a]	1.268 (1.070-1.502)	.006

CI, Confidence interval; *EL,* endoleak; *FU,* follow-up; *HR,* hazard ratio.
[a]Per unit increase in endoleak.

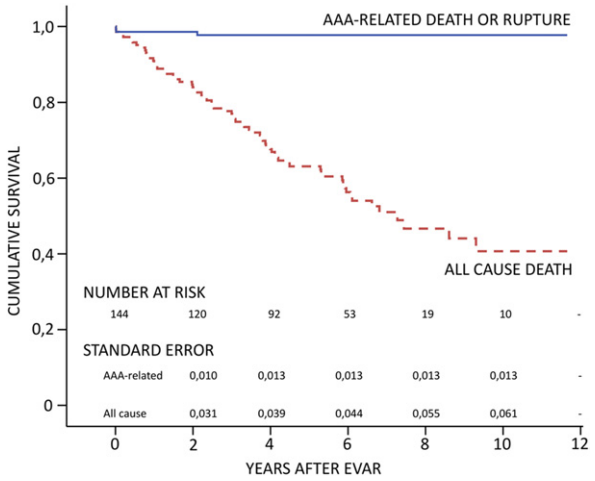

Fig 3. Kaplan-Meier estimates are shown for overall (*dashed red line*) and abdominal aortic aneurysm (*AAA*)-related survival (*solid blue line*). *EVAR*, Endovascular aneurysm repair.

Long-term survival. Sixty-eight patients died during follow-up. Estimated survival after AAA repair was 6.8 years (95% CI, 6.1-7.5 years; Fig 3). Only three AAA-related deaths occurred: two perioperative deaths and one of infection, as mentioned. The most frequent cause of death was cancer-related, occurring in 29 of 68 (42.6%), followed by myocardial infarction in 10 (14.7%) and stroke in six (8.8%). Overall survival of patients treated urgently was similar to those treated electively, even when 30-day mortality was included (log-rank $P = .117$).

DISCUSSION

Despite evidence of an early advantage over open repair, doubt remains regarding the durability of EVAR.[6,7] More than two decades after the introduction of the technique, solid long-term results remain scarce and limited by the permanent introduction of new devices. The Gore Excluder endoprosthesis was the preferred device in our institution for many years and was used in >80% of patients. Aside from the introduction of the LP design in 2004 and recent developments in the deployment system, this device has been virtually unchanged since its introduction. Therefore, our study provides valuable long-term information with current clinical applicability.

Technical success and early results. Intraoperative adjunct procedures were required in 6.2% of patients to achieve or enhance proximal fixation and seal. Another 2.8% were found to have partial occlusion of a renal artery. These complications were fundamentally associated with inaccurate deployment. This may have led to a bias in patient selection toward use of this device in longer proximal necks. It may also explain why, unlike previously reported,[11] only neck diameter was associated with intraoperative type Ia endoleak.

In-hospital and 30-day mortality was remarkably low (1.4%) for a cohort in which 62% was assessed as American Society of Anesthesiologists classes 3 and 4, including urgent operations. These outcomes compare favorably with those of randomized EVAR trials and may result from selection bias.[5,12-14]

Clinical success. Midterm clinical results using the Excluder endoprosthesis have been well characterized,[15-20] but long-term clinical outcomes (>4 years) are scarce. Recently, Maleux et al[21] published their 10-year experience with the Excluder device. Overall, their results are similar to ours in regard to early success and late complications. However, their numbers were smaller (n = 121) and included patients with isolated iliac aneurysms (13%), mycotic (5%), and pseudoaneurysms (1.7%), which could bias the overall analysis. Hogg et al[22] analyzed the long-term sac behavior with the Excluder device, but outcomes were compromised by a significant number of patients lost during follow-up: after 1 year, only about two-thirds of patients were available for analysis. To adequately interpret long-term results, completeness of follow-up is an essential prerequisite that our study fulfills.

Long-term results were also recently published for the Talent endoprosthesis.[23] They reported similar primary clinical success rate of 64% at 5 years but a much higher estimated AAA-related mortality of 8% at 7 years and four postimplantation ruptures at a mean of 40 months, all associated with graft migration and type I endoleak. Another recent publication reported long-term outcomes using the Zenith endoprosthesis in 143 elective patients, of whom four had incomplete imaging data.[24] They found similar intervention-free survival at 5 years of 77%, but six postimplantation ruptures were noted at a mean follow-up of 66 months.

In our study, clinical success was highly dependent on sac growth. As has been previously reported, the generation of the implanted device influenced sac growth—OD grafts increased the risk of sac growth significantly.[15,18,19,25] The large study by Schanzer et al[22] reported an overall 41% risk of sac growth at 5 years; however, no device-specific data were available. In that study, the chance of growth was greater from 2004 to 2008, after introduction of the LP design.

Our results differ, showing a markedly reduced proportion of patients with sac growth after 2004. Accordingly, Hogg et al reported 14.8% sac growth at 4 years for 301 patients treated with the LP. In our study, volume measurements may have introduced a bias, because a larger proportion of patients are classified as having postimplantation sac growth using volume compared with diameter. Because postoperative aneurysm sac volume has been shown to better reflect the efficacy of treatment,[26,27] we believe it provides a more reliable estimate. Importantly, a clear motif for sac growth (eg, type I endoleak or migration) was found in all but one patient treated with the LP endoprosthesis. Conversely, no rupture or AAA-related death was observed in 13 patients with endotension treated conservatively.

Over time, neck dilatation occurred in one-third of patients and was associated with the degree of oversizing. In patients with the longest follow-up, neck dilatation was very common, reflecting the tendency for continued neck dilatation over time when self-expanding nitinol grafts are used. Our data support that long-term dilatation beyond device diameter is rare, and when present, is associated with progression of aneurysmal disease.[28] Reports on neck dilatation differ significantly, perhaps due to institutional policies on oversizing and device-related characteristics. However, others have reported higher dilatation rates in devices with suprarenal fixation, suggesting that as an additional factor promoting dilatation.[29-32]

Our cohort had few occlusive complications, occurring exclusively in patients with narrow aortic bifurcations, a known risk factor for occlusion.[33] Data for European Collaborators on Stent-Graft Techniques for Aortic Aneurysm Repair (EUROSTAR) and single-center reports further support the good performance of this device in adverse iliac anatomy,[33-35] but this was not directly evaluated in our population.

Postimplantation rupture has been recognized as the paradigm of EVAR failure: a report from a large cohort of Medicare beneficiaries noted a rupture risk of 1.8% at 4 years.[36] Subsequently, Wyss et al[6] analyzed postimplantation ruptures from EVAR trial patients and found a rupture rate of 0.7/100 person-years. They suggested this could explain the loss of early benefit for EVAR compared with open repair. Two ruptures occurred in the EVAR trials after implantation of the Excluder device. In the Veterans Affairs Open versus Endovascular Repair (OVER) trial, with 327 (37.1%) Excluder devices implanted and a mean follow-up of 1.8 years, no late ruptures were documented.[5] The same was observed after a median of 6.0 years in the Dutch Randomised Endovascular Aneurysm Management (DREAM) trial (no device-specific data).[4] Several observational studies analyzing the long-term performance of the Excluder device have reported no late ruptures.[21,22,37-39] One patient in our series suffered rupture, although this was caused primarily by endograft infection.

After EVAR, eGFR rates remained stable or slowly declined in most patients, despite an intensive CTA surveillance protocol, with a mean decline of 4.8 mL/min/1.73 m^2 over 5 years. We observed transient worsening of eGFR after EVAR and recovery to near-baseline levels at the end of follow-up. Greenberg et al[40] previously described this U-shaped curve, and we confirm their observation over longer follow-up.

Long-term survival. Our expected survival at 6 years was close to 50%, which is below the expected rate for the same period for the Comparison of Endovascular Aneurysm Repair with Open Repair in Patients with Abdominal Aortic Aneurysm (EVAR-1) and DREAM trials[3,4] but above the expected survival from the United Kingdom Endovascular Aneurysm Repair 2 (EVAR-2) trial.[2] We report an "every-day" population, perhaps resulting in a more realistic expectation for clinical practice. Interestingly, most deaths were cancer-related. To further improve the long-term survival of AAA patients, a more thorough and multidisciplinary approach to comorbidities and risk is desirable.

Limitations. The observational design and single-center cohort, with inherent selection bias, limit this study; however, unlike most long-term studies, we provide complete follow-up, including cause of death for all patients. As such, we avoid omitted complications that never reach hospital care or get treatment elsewhere. We also acknowledge the relatively small population size.

CONCLUSIONS

This study offers a thorough analysis on clinical outcome and morphologic aneurysm changes up to 11 years after EVAR using the Excluder endoprosthesis. AAA-related mortality was exceptionally low, although clinical success was compromised by a large proportion of sac growth in patients treated with the OD generation endografts. Despite continued need for surveillance and intervention, these results provide reassurance for AAA treatment with a currently commercialized endoprosthesis.

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AUTHOR CONTRIBUTIONS

Conception and design: FB, MV, HV
Analysis and interpretation: FB, AJ, ER, SR, JH, AM, MV, HV
Data collection: FB, AJ
Writing the article: FB, ER, SR
Critical revision of the article: ER, JH, AM, MV, HV

Final approval of the article: FB, AJ, ER, SR, JH, AM, MV, HV
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